

that these organisms play a major role in the pathogenesis of tropical sprue in southern Africa. The pathogenesis of tropical sprue in South Africa is unknown, but probably differs from that postulated for sprue in India, Puerto Rico, and the West Indies.

The significance of the greater number of *Haemophilus* species isolated from sprue patients than controls, and the greater number of  $\beta$ -haemolytic streptococci isolated from controls than sprue patients, is unknown. Although the greatest care was taken to prevent contamination of specimens with oral flora, the possibility that such contamination could have occurred cannot be ruled out. Specimens were collected by methods similar to those reported previously by workers in the USA,<sup>1-7</sup> so that our results are probably comparable in this respect. In addition, subtle changes in intestinal flora, which would have remained undetected by the methods used to quantitate and identify intestinal microflora, may play a role in the pathogenesis of tropical sprue in our patients.

Further evaluation of the pathogenesis of tropical sprue in Natal should include studies of the influence of specific anti-aerobic (e.g. aminoglycoside) and anti-anaerobic (e.g. nitro-imidazole) agents on the symptomatology and intestinal bacteriology of tropical sprue patients. Quantitative bacteriological studies of specimens from other gastro-

intestinal sites (stomach, upper jejunum, ileum, faeces) should also be carried out.

This study was supported by grants from the Group Chairman's Fund of the Anglo-American Corporation, and the South African Medical Research Council. Ms Nancy L. Chambers is thanked for help with statistical analyses.

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## Pneumoperitoneum in the Neonate

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### SUMMARY

Pneumoperitoneum in the newborn child is not necessarily diagnostic of visceral perforation. If perforation can be excluded, these patients can be spared the risks of an operation.

*S. Afr. med. J.*, **57**, 1083 (1980).

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Date received: 28 June 1979.

Pneumoperitoneum in the neonate has long been regarded as diagnostic of visceral perforation. However, an increasing number of cases have been reported in which no visceral perforation has been shown and in which pneumoperitoneum has accompanied respiratory distress, especially when assisted ventilation has been applied.

Two cases of pneumoperitoneum are presented, with a brief review of the world literature. The investigation and treatment of these patients is discussed.

### CASE REPORTS

#### Case 1

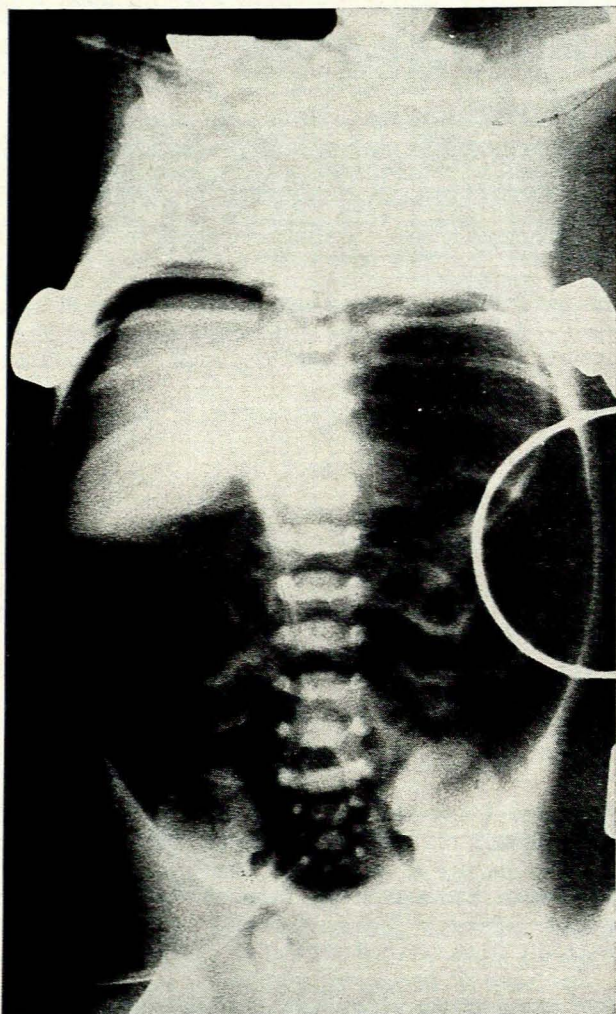
A 1420 g male infant was delivered prematurely in a country hospital. During resuscitation he was intubated and ventilated with intermittent positive-pressure respira-



tion (IPPR). He responded well and spontaneous respiration was established, upon which he was extubated.

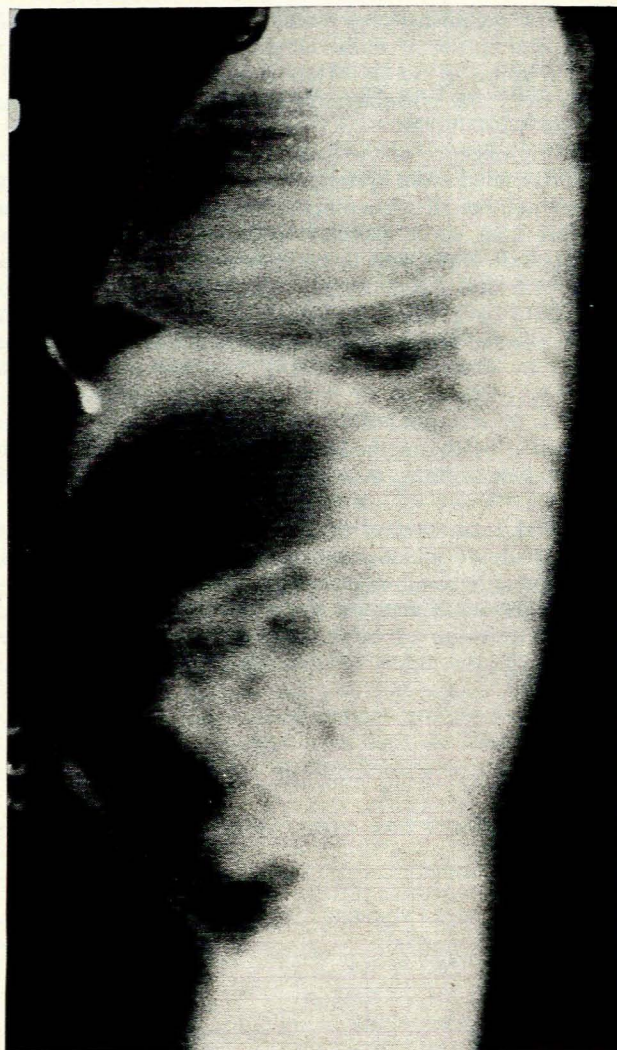
Some hours later the patient developed signs of respiratory distress, and oxygen (2 l/min) was administered through a nasal catheter. The patient was then transferred to Tygerberg Hospital.

On arrival he had peripheral cyanosis, was grunting and was tachypnoeic, with marked rib retraction. The abdomen was markedly distended and tympanitic, and scrotal swelling was noted. Bowel sounds were absent. Abdominal and chest radiographs showed mild hyaline membrane disease and a marked pneumoperitoneum (Figs 1 and 2). There were no air/fluid levels or signs of free fluid in the abdomen.



**Fig. 1. Case 1. Anteroposterior radiograph showing gross pneumoperitoneum and features of hyaline membrane disease.**

Because of the patient's poor condition it was decided to treat him conservatively with nasogastric drainage, total parenteral nutrition and antibiotics. IPPR was necessary to maintain adequate gas exchange.



**Fig. 2. Case 1. Lateral radiograph showing the same features.**

Over the next 12 days the patient's condition remained stable, and there was radiological evidence that the pneumoperitoneum had resolved after 48 hours (Fig. 3). It was necessary to continue assisted respiration for 12 days before he could be weaned from the respirator.

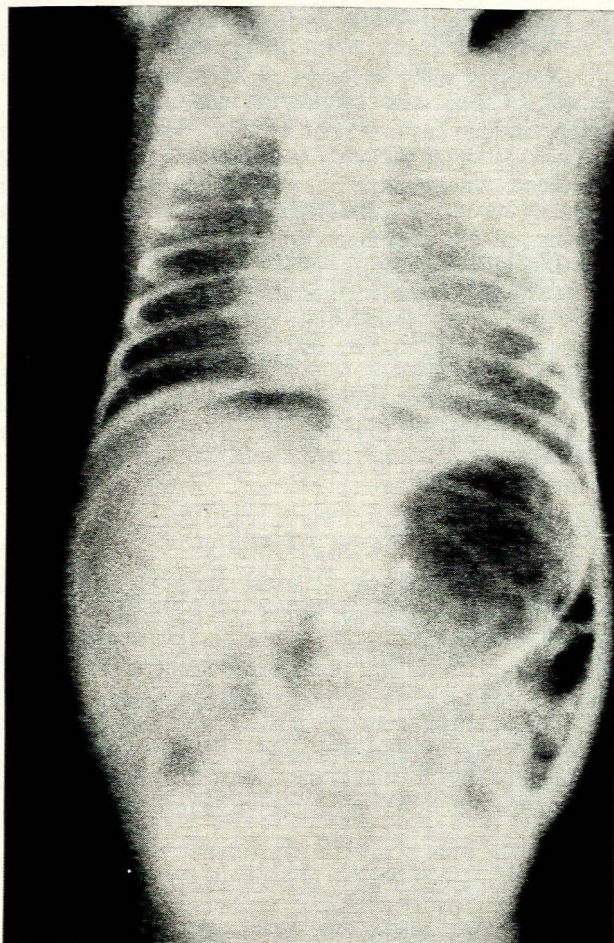
After 15 days of total parenteral nutrition nasogastric feeds were commenced, the patient being given sterile water initially and then a standard humanized milk formula. No further problems were encountered, and he was discharged from the unit 27 days after admission.

## Case 2

An 870 g male infant was born by spontaneous vaginal delivery after the premature onset of labour.

On admission to the unit he had mild peripheral cyanosis with tachypnoea, grunting and rib retraction. A chest radiograph showed perihilar streaking, widening of the rib spaces and diaphragmatic flattening.





**Fig. 3. Case 1. The pneumoperitoneum has resolved completely 3 days later, and the chest looks more normal.**

He was placed in a constant negative-pressure respirator with a pressure of  $-5$  cm  $H_2O$  in 30%  $O_2$ . Total parenteral nutrition was commenced via an umbilical venous catheter.

The clinical picture improved and by day 3 his blood gas values were normal when he was breathing room air. Constant negative pressure was maintained.

On day 7 he had mild abdominal distension, a cyanosed left arm and a gangrenous left little toe. There were no other signs of embolus formation. The umbilical catheter was removed and intravenous fluid therapy was commenced through a peripheral vein. An erect radiograph of the abdomen showed a massive pneumoperitoneum. There was no sign of free fluid in the abdomen. Intravenous nutrition and intravenous antibiotics were continued, with constant nasogastric suction, but the patient's condition deteriorated and he died on the 10th day, with generalized haemorrhage from the trachea, mucous membranes and venepuncture sites.

At autopsy there were signs of disseminated intravascular coagulation, but no emboli were found in the viscera. Two ileal perforations were present, with a localized peritonitis, and there was evidence of localized

necrotizing enterocolitis on microscopic examination of the region of the perforations. Histological examination of the lungs revealed areas of atelectasis.

## DISCUSSION

Pneumoperitoneum in the newborn has long been accepted as evidence of perforation of an abdominal viscus and an indication for immediate surgical intervention.<sup>1-3</sup> In 1966 Mestel *et al.*<sup>3</sup> presented 32 selected cases of pneumoperitoneum which they defined as 'radiological evidence of rupture of an air-containing viscus with resultant gross soiling of the peritoneal cavity'. Thirty of these had gastro-intestinal perforations at sites ranging from the stomach to the rectum, but no perforations could be found in the other 2, and there was no mention of respiratory distress in any. The radiological signs were discussed, but there was no reference to air/fluid levels or free fluid in the abdomen. The authors stated that 'if free air is seen, barium studies are unnecessary'. They felt that all patients with pneumoperitoneum should undergo exploratory surgery.

It is now clear that in a few cases pneumoperitoneum in the newborn is not due to rupture of abdominal viscera,<sup>4,5</sup> although the latter is certainly the cause in the majority of cases. In 1956 Porter<sup>4</sup> reported an otherwise normal infant with pneumoperitoneum. No perforation was found at laparotomy, there was no respiratory distress or pneumothorax, and the patient recovered after the operation. In 1970 Leiniger *et al.*<sup>5</sup> reported a neonate who developed right tension pneumothorax and pneumoperitoneum shortly after birth. The pneumothorax was drained, with resolution of the pneumoperitoneum.

In 1973 Leonidas<sup>6</sup> reported the case of an 800 g infant who developed pneumoperitoneum following severe respiratory distress requiring assisted ventilation. There was no evidence of pneumothorax or pneumomediastinum. Autopsy revealed no evidence of gastro-intestinal tract perforation, and examination of the lungs showed acute confluent bronchopneumonia. In 1974 Brown and Keenan<sup>7</sup> reported an 880 g neonate who required assisted ventilation after premature delivery. This infant developed pneumoperitoneum some hours after birth, with no radiological evidence of pneumomediastinum or pneumothorax. Autopsy revealed hyaline membrane disease and microscopic evidence of interstitial emphysema. There was no evidence of bowel perforation or inflammatory bowel disease.

In 1975 Leonidas *et al.*<sup>8</sup> presented a series of 222 infants who underwent IPPR in the course of treatment. Of these 9 developed pneumoperitoneum, 4 with evidence of a perforation at laparotomy or at autopsy. The authors found that 'the presence or absence of pulmonary air leaks correlated poorly with the presence or absence of a perforation'. Two of the patients who had visceral perforations showed air/fluid levels on abdominal radiographs, while the other 2 did not — air/fluid levels were never seen in patients who did not have visceral perforations. Contrast medium injected into the stomach in one patient revealed an ileal perforation. Shija<sup>9</sup> reported a case of pneumoperitoneum in a newborn infant which he



presumed to be a result of 'quiet perforation in some part of the gastro-intestinal tract'. No mention is made of radiological evidence of free fluid, and apparently the infant was not distressed. It did well on conservative non-operative treatment. Mangurten *et al.*<sup>10</sup> reported 2 extremely premature infants who developed pneumoperitoneum while undergoing IPPR with facemasks. Both were presumed to have sustained rupture of the gastro-intestinal tract owing to overdistension of the stomach in the course of the ventilatory procedures. In one case the leak was discovered by the injection of a water-soluble contrast medium through the nasogastric tube. No free fluid was seen on abdominal radiographs in either case. Both patients survived on conservative treatment with nasogastric suction, intravenous fluids, antibiotics and ventilation by endotracheal tube.

The pathogenesis of pneumoperitoneum in the neonate has been the subject of some debate. In 1944 Macklin and Macklin<sup>11</sup> described the development of pulmonary interstitial emphysema in patients with raised intra-alveolar pressures and atelectasis. Alveolar rupture resulted in air dissecting along the peribronchiolar blood vessels to form a pneumomediastinum and/or surgical emphysema. Pleural rupture then led to the formation of a pneumothorax. Donahoe *et al.*<sup>12</sup> showed experimentally in rats with artificially induced pneumothorax that dissection of air took place through the para-oesophageal and para-aortic spaces in the diaphragm. The pneumoretroperitoneum then ruptured through the mesentery to form a pneumoperitoneum.

Brown and Keenan,<sup>7</sup> however, felt that air may pass from the chest to the peritoneum retrograde along the lymphatic system, since in their opinion the air seen on microscopic examination in their patient with interstitial emphysema was located in the peribronchiolar lymphatics.

Radiological signs are the most important diagnostic features of pneumoperitoneum, consisting of (a) the presence of air in the peritoneum, especially in the sub-diaphragmatic space; (b) evidence of free fluid or air/fluid levels, which indicate that a perforation has taken place — their absence on an erect abdominal radiograph, however, does not exclude perforation; (c) the bowel wall pattern may show evidence of necrotizing enterocolitis, which may be present from the 1st day of

life; and (d) water-soluble contrast medium may be used to demonstrate a leak if the diagnosis is still uncertain, but great care must be taken to avoid dehydration, and the risk of peritonitis cannot be ignored.

If a decision is made not to undertake an operation, treatment should include: (a) nasogastric suction; (b) intravenous maintenance of fluid balance and nutrition; (c) broad-spectrum antibiotics; (d) percutaneous decompression of the pneumoperitoneum, which may reduce intra-abdominal pressure and improve diaphragmatic movement (a simple method is to insert, using aseptic technique, a soft intravenous cannula connected to an underwater drain — this will help to detect peritoneal soiling if a perforation is present, and drainage of any associated pneumothorax or pneumomediastinum can lead to rapid resolution of pneumoperitoneum); and (e) assisted diaphragmatic splinting may contribute markedly to respiratory complications.

## CONCLUSION

While the vast majority of cases of pneumoperitoneum are certainly surgical in origin, a few cases are not. Pneumoperitoneum *per se* can no longer be presumed to be diagnostic of visceral perforation. If there is reasonable certainty that the air is pulmonary in origin, an often very sick patient may be spared the added stress of hazardous and unnecessary surgical intervention. This is no easy decision to make, but no effort should be spared to demonstrate bowel perforation before operating.

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